REMARKS

Favorable reconsideration is respectfully requested.

The claims are 5 and 6.

The above amendment is responsive to the rejections under 35 USC § 112 in Official Action paragraphs 5 to 10. The method of curing is no longer claimed.

Claims 5 and 6 are rejected under 35 USC § 103(a) as being unpatentable over Morgan et al. in view of NCI-Antioxidant Cancer Prevention (NCI) and further in view of Buchter-Larsen et al. and Behrend et al.

In the Official Action dated August 6, 2008, it was stated that "Applicants have not presented any evidence that APP functions in a manner distinct from that which is known in the art to be associated with its precursor 1,5-anhydro-D-fructose" (see page 4, lines 3-5). However, it is detailed at pages 6 to 8 of the specification and is clearly set forth in the Rule 132 Declaration of Dr. Abeyama dated May 10, 2008, that APP exhibits induction of apoptosis. This is in contrast to 1,5-anhydro-D-fructose which inhibits apoptosis.

Further, present claims 5 and 6 recite apoptosis induction as suggested in item 6 on page 4 of the previous Office Action dated August 6, 2008.

Turning to the cited references:

1. Morgan et al. merely teaches that ascopyrone P is known to function as a good antioxidant however, this would not lead one of ordinary skill in the art to recognize its use in the presently recited method, namely, treating patients to induce apoptosis of the tumor cells, thereby killing the tumor cells directly.

2. NCI on page 1, under the heading "Can antioxidants prevent cancer?" states that:

> Considerable laboratory evidence from chemical, cell culture, and animal studies indicates that antioxidants may slow or possibly prevent the development of cancer. However, information from recent clinical trials is less clear. In recent years, large-scale, randomized clinical trials reached inconsistent conclusions (emphasis added).

The contention of the rejection that antioxidants can be used to treat or prevent cancer thus goes well beyond what is actually taught by NCI, and NCI certainly does not disclose or suggest that it is possible to induce apoptosis of tumor cells, thereby killing the tumor cells directly, with any antioxidant, no less ascopyrone.

Also, as pointed out in the response dated November 15, 2007 at page 5, the number of antioxidants which show an antitumor effect by themselves is very limited.

3. Buchter-Larsen et al. merely teaches the preparation and use of 1,5-D-anhydrofructose (a precursor to ascopyrone) as a water soluble antioxidant. However, as discussed above, different antioxidants exhibit different effects in the treatment of cancer.

4. Behrend et al. teaches that ROS (reactive oxygen species), e.g., free radicals, have been implicated in mediating loss of growth control i.e., tumorigenic activity and have also been implicated in apoptosis, the latter being regarded as being anti-tumorigenic. See Abstract and on page 1442.

Behrend refers to this as the "two faced" character of free radicals. Again, see Abstract.

Therefore, one seeking to kill tumor cells directly by apoptosis would not be taught by Behrend to eliminate ROS with antioxidants but rather to avoid them.

In sum, the cited references, alone or combined, completely fail to teach or suggest that ascopyrone can be employed to induce apoptosis in tumor cells, thereby killing the tumor cells directly.

For the foregoing reasons, it is apparent that the rejection on prior art is untenable and should be withdrawn.

No further issues remaining, allowance of this application is respectfully requested.

If the Examiner has any comments or proposals for expediting prosecution, please contact undersigned at the telephone number below.

The Commissioner is authorized to charge any deficiency or to credit any overpayment associated with this communication to Deposit Account No. 23-0975, with the EXCEPTION of deficiencies in fees for multiple dependent claims in new applications.

Respectfully submitted,

Ikurou MARUYAMA et al.

By: _____

Matthew M. Jacob
Registration No. 25,154
Attorney for Applicants

MJ/lkd
Washington, D.C. 20005-1503
Telephone (202) 721-8200
Facsimile (202) 721-8250
July 1, 2009